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Follicular Dendritic Cell Sarcoma Mimicking Diffuse Large Cell Lymphoma: A Case Report

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Follicular dendritic cell sarcomas (FDCSs) are rare tumors arising from follicular dendritic cells in lymphoid tissue. Fewer than 20 cases have been described in the English-language literature. We describe the second case of an FDCS with primary liver involvement. The initial diagnosis was lymphoma, and appropriate treatment was prescribed. After the initial treatment failed, additional biopsy samples were obtained. Standard pathologic analysis and immunophenotyping for a panel of monoclonal antibodies were performed on formalin-fixed paraffin-embedded tissue and frozen sections. The pathologic findings were consistent with FDCS, and the specimens showed some of the characteristic pathologic features suggestive of this tumor, including multinucleation and a spindle pleomorphic morphology. The tumor cells were positive for S-100 protein, CD45, CD14, and vimentin. Because of its morphological characteristics, FDCS can be confused with other neoplastic entities, such as lymphomas and other solid tumors. Am. J. Hematol. 55:148–155, 1997. © 1997 Wiley-Liss, Inc.

Key words: liver; neoplasm; treatment

INTRODUCTION

Follicular dendritic cells (FDCs) are antigen-presenting reticular cells in the germinal center of lymph nodes in close contact with B lymphocytes [1,2]. Morphologically, they are spindle-shaped multinucleate cells with cytoplasmic dendritic projections and desmosomal attachments [3–5]. Antigen expression by FDCs is usually variable [6] and includes both specific (R4/23, Ki-M4) [7] and nonspecific surface determinants [3,8–11]. The proximity of FDCs to nodal lymphocytes and macrophages has caused difficulty in defining a characteristic immunophenotypic profile. FDCs are considered accessory cells of the immune system, presenting antigen-antibody complexes to B lymphocytes [12,13] and possibly participating in B-cell proliferation and differentiation [14].

Benign and neoplastic proliferations of FDCs are rare. Inflammatory pseudotumor (IPT) represents a benign clustering of FDCs and is seen in lymph nodes [15] and in extranodal locations [16,17]. A malignant proliferation of FDCs is known as ''FDC sarcoma'' (FDCS). These tumors are rare, and fewer than 20 cases have been reported in the English-language literature [18–27]. Conceivably, the actual incidence of FDCS is underestimated because of its clinical and pathologic resemblance to lymphoproliferative disorders. The usual clinical presentation is that of asymptomatic cervical or axillary (or both) lymphadenopathy.

Herein, we describe the second reported case of an FDCS that involved primarily the liver and spleen. The challenges in diagnosis and the clinical behavior abstracted from the available literature are discussed.

CASE HISTORY

We evaluated a 64-year-old man in whom the original diagnosis was a diffuse large cell lymphoma. The patient presented to his local physician in May 1995 complaining of diffuse upper abdominal discomfort, night sweats, and fever. Computed tomographic (CT) examination of the abdomen showed multiple liver lesions. The results of laparoscopically guided biopsies were reported to be

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consistent with a diffuse large cell lymphoma. Pathologic samples submitted to our institution showed that the tumor cells expressed CD45, consistent with large cell lymphoma. Bone marrow biopsy specimens failed to disclose pathologic involvement.

The patient had no significant past medical history. In 1986, he was evaluated at our institution, at which time he complained of left upper quadrant pain. The results of the physical examination and laboratory tests were all within normal limits. No enlarged, palpable peripheral lymph nodes were detected. The liver and spleen were both normal. The results of ultrasonographic examination of the liver and spleen and abdominal CT in 1986 were reported as normal.

The physical examination findings in May 1995 were entirely normal. He did not have hepatosplenomegaly or lymphadenopathy. The following laboratory results were obtained: hemoglobin, 12.6 g/dL; white blood cells, 7.5×10^9/L; platelets, 366×10^9/L; and differential count, normal. The following chemistry laboratory values were within normal limits: lactate dehydrogenase, 174 U/L (normal, 112–257 U/L); serum creatinine, 1.1 mg/dL (normal, 0.8–1.2 mg/dL); serum calcium, 9.3 mg/dL (normal, 8.9–10.1 mg/dL); total protein, 6.9 g/dL (normal, 6.3–7.9 g/dL); albumin, 3.8 g/dL (normal, 3.5–5.0 g/dL); aspartate aminotransferase, 19 U/L (normal, 12–31 U/L); total bilirubin, 0.6 mg/dL (normal, 0.1–1.1 mg/dL); direct bilirubin, 0.1 mg/dL (normal, 0.0–0.3 mg/dL). His alkaline phosphatase was slightly increased at 295 U/L (normal, 98–251 U/L). Peripheral blood smear analysis did not reveal gross evidence of circulating neoplastic cells. Chest radiographic findings were normal. Abdominal CT scan showed disease localized to the liver, spleen, and retroperitoneal lymph nodes (Figs. 1 and 2). Multiple hypodense lesions were seen in the liver and spleen parenchyma, and some of them appeared to have a necrotic center.

The patient had chemotherapy with six cycles of CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone). No major complications occurred because of the treatment. The fever and night sweats resolved, but the right upper quadrant discomfort persisted. Because of a limited partial response to treatment, it was decided to proceed with a diagnostic splenectomy and liver biopsy. Intraoperatively, an enlarged spleen with multiple peritoneal implants was found, suggestive of involvement by a lymphomatous process. Multiple liver biopsy samples were obtained of the affected areas.

After surgical exploration, splenic vein thrombosis developed and required long-term anticoagulation with oral agents. The patient elected not to undergo further chemotherapy and has been observed since that time. There has been a slowly progressive increase in the size of the retroperitoneal lymph nodes and several enlarged mediastinal lymph nodes have been detected.

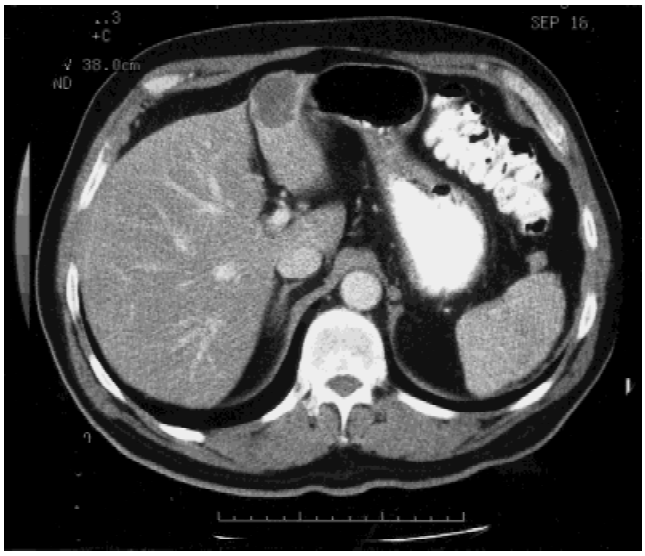

Fig. 1. Abdominal computed tomographic scan showing the parenchymal lesion in the lateral segment of the liver.

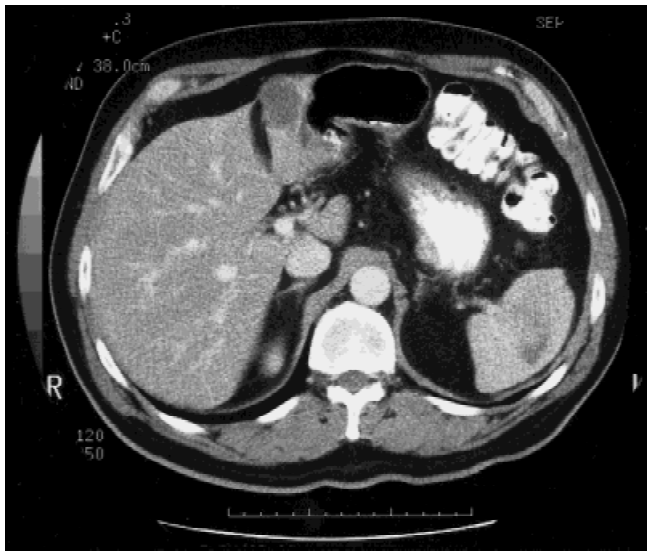

Fig. 2. Abdominal computed tomographic scan showing the hypodense lesion in the spleen.

MATERIALS AND METHODS

A standard pathologic analysis of the specimens was performed and reviewed by several hematopathologists. The frozen sections and formalin-fixed paraffin-embedded tissue was immunostained for CD45 (LCA), S-100 protein, CD14, CD20, CD21, CD22, kappa, lambda, CD2, CD3, CD5, CD7, CD15, CD30, CD35, CD23, bcl-2 protein, AE1/AE3, CAM 5.2, CD43, MPO, CD31, factor VIII, actin, desmin, and HMB-45 (Table I).

Using Southern blot analysis, gene rearrangement studies were conducted on fresh frozen tissue to detect clonal rearrangements of the immunoglobulin genes mu

TABLE I. Immunophenotyping Antibodies

Antibody	Specificity	Commercial source
CD45 (LCA)	Leukocyte common antigen	Dako (Carpinteria, CA)
S-100	Melanoma	HSC (Toronto, Canada)
CD14	Myeloid	Dako
CD20	Pan-B cell	Dako
CD22	Pan-B cell	Becton Dickinson (San Jose, CA)
Kappa	Immunoglobulin	Becton Dickinson
Lambda	Immunoglobulin	Becton Dickinson
CD2 (Leu-5b)	Pan-T cell	Becton Dickinson
CD3 (Leu-4)	Pan-T cell	Becton Dickinson
CD5 (Leu-1)	Pan-T cell	Becton Dickinson
CD7 (Leu-9)	Pan-T cell	Becton Dickinson
CD15 (Leu-M1)	Reed-Sternberg cells	Becton Dickinson
CD30	Reed-Sternberg cells	Dako
CD21 (IF 8)	Dendritic cells	Dako
CD 35 (Ber Mac DRC)	Dendritic cells	Dako
CD23	B cells	Dako
bcl-2	Mantle zone B cells	Dako
AE1/AE3	Keratin	Boehringer Mannheim (Indianapolis, IN)
CAM 5.2	Keratin	Becton Dickinson
CD43 (Leu-22)	Myeloid, T cells	Becton Dickinson
MPO	Myeloid	Dako
CD31	Endothelial	Dako
Factor VIII	Endothelial	Dako
Actin (HHF 35)	Muscle-specific	Dako
Desmin	Muscle-specific	Dako
HMB-45	Melanoma	Dako

and kappa and the T-cell receptor genes beta and gamma.

In situ hybridization studies to detect Epstein-Barr virus mRNA were done with the EBER probe.

RESULTS

The pathologic specimens consisted of portions of the liver and spleen (spleen: weight, 275 g; size, 15 by 9.5 by 4.5 cm). The sections of spleen and liver revealed similar partial infiltration by an abnormal, vaguely nodular process containing a predominance of spindle cells with admixed small lymphocytes. The spindle cells displayed large nuclei with marginated chromatin and distinct, occasionally large, nucleoli. Some of the spindled cells displayed polylobate or bilobed nuclei (Figs. 3 and 4).

Immunostains showed that the tumor cells were weakly reactive for CD45 (LCA), S-100 protein, CD14, and vimentin. We were unable to demonstrate conclusively reactivity of the neoplastic cells for other markers, including B-lineage markers (CD20, CD22, kappa, lambda), T-lineage markers (CD2, CD3, CD5, CD7), Hodgkin's-associated markers (CD15, CD30), other lymphoid markers (CD23, bcl-2 protein), antibodies to keratin (AE1/AE3, CAM 5.2), myeloid markers (CD43, MPO), vascular markers (CD31, factor VIII), muscle-specific markers (actin, desmin), or HMB-45. Immunostains for CD21 and CD35 were not found to be reactive. Molecular genetic studies (fresh frozen tissue) showed no evidence of clonal rearrangements of immunoglobulin genes (mu, kappa) or T-cell receptor genes (beta, gamma). Special stains for acid-fast bacilli (auramine-rhodamine) and fungi (GMS) were negative. In situ hybridization studies to detect the Epstein-Barr virus were negative. The final pathologic diagnosis was that of an unusually pleomorphic FDCS.

DISCUSSION

Primary reticular spindle cell neoplasms of lymphoid tissue include FDCS and tumors of the interdigitating reticulum cell (IDRC) lineage [19]. Both are rare disorders with only 17 well-documented cases of FDCS in the literature (Table II) [18–26]. Thus, information about their biologic behavior and natural history remains inadequate. Patients with FDCS usually present with cervical or axillary lymphadenopathy. The morphological analysis of FDCS reveals an abnormal collection of bilobated spindle cells that replace the normal lymph node architecture [23]. The neoplastic cells are morphologically and immunophenotypically similar to normal FDCs [23,24,27]. Electron microscopic studies show that the cells have the long cytoplasmic projections and desmosomal bodies normally found in FDCs. On the one hand, the neoplastic cells in FDCS generally express HLA-DR and complement receptors, including CD21, but they lack the usual B- and T-cell markers (Table III). On the other hand, the expression of S-100 protein, leukocyte common antigen (CD45), and vimentin is variable [21,24,25]. Molecular genetic studies for clonal T-cell receptor or immunoglobulin gene rearrangements have been nondiagnostic for a monoclonal cell population [19,21].

The overall clinical course and treatment outcome of 17 previously reported cases of FDCS are summarized in Table I. In most patients, the disease had a prolonged and indolent clinical course [19,20,22]. Of the 17 reported cases of FDCS, 13 patients presented with lymphadenopathy (8, cervical; 3, axillary; 1, mesenteric; and 1, tonsillar) and 4 had an extranodal presentation (1, liver; 1, palate; 1, small bowel; and 1, pancreas). Eleven patients received treatment with surgical excision alone, of whom seven were alive and free of disease at the time of the report. Three patients had radiation therapy in addition to surgical excision and achieved complete remission. Of the two patients who received chemotherapy, one achieved complete remission and the other was lost to follow-up. After a median follow-up of 1 year (range, 4 to 90 months), 11 patients were alive and disease-free, 1 had died of disease, and no follow-up information was

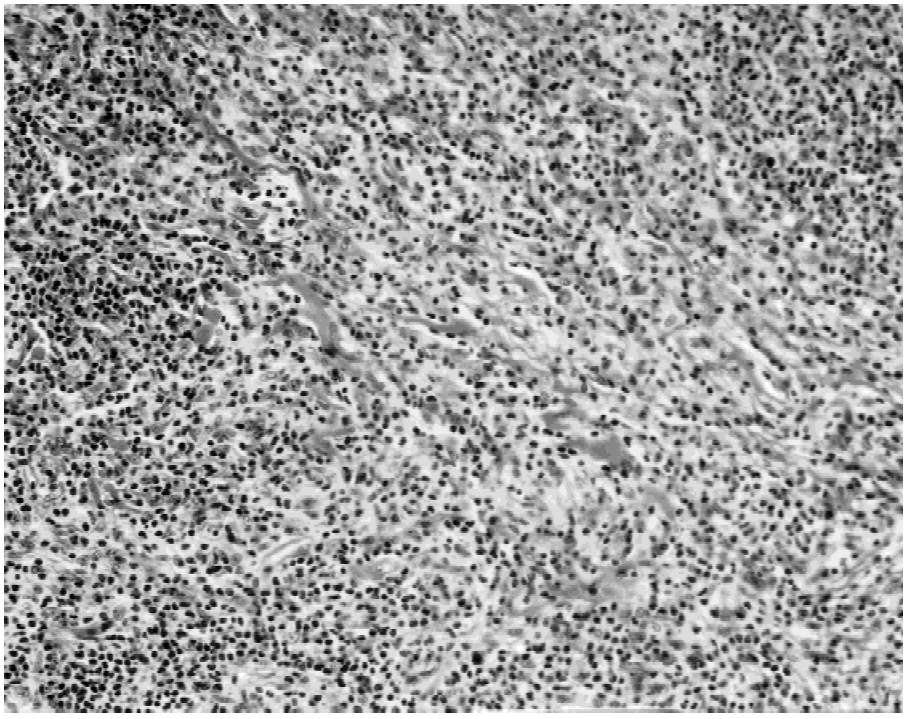

Fig. 3. The neoplasm has a diffuse architectural pattern. (Hematoxylin-eosin, ×200.)

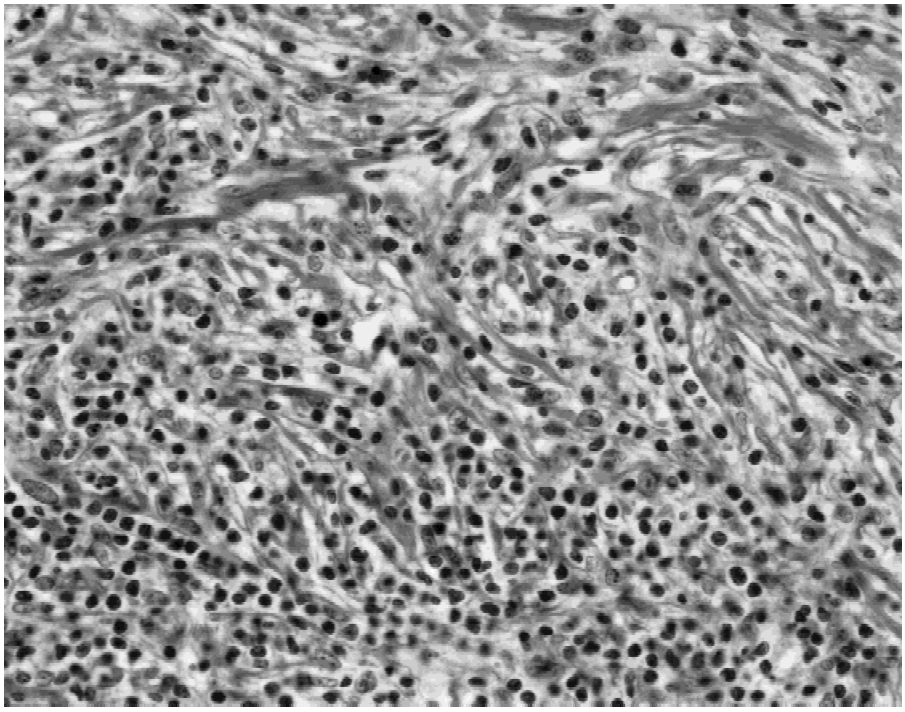

Fig. 4. Some of the neoplastic cells are spindle cells. (Hematoxylin-eosin, ×400.)

available for the other five. Longer follow-up time and more experience are needed to define further the natural history and the role of different treatment modalities.

Morphologic analysis in our patient revealed typical spindle cells with poly- or bilobed nuclei. Similar to some, but not all, of the previously reported cases, immunostaining for CD45 was weakly reactive. The neoplastic cells in our patient also expressed a myeloid antigen (CD14), which when coexpressed with CD45 is believed to suggest a hematopoietic origin for the tumor [9]. Staining for S-100 protein, an antigen usually expressed in IDRCs, was weakly positive in our patient, as in other previously reported ones [19,20]. In our case, the staining for CD21 and CD35 was not reactive. Unfortunately, no glutaraldehyde-fixed tissue was available for electron microscopic study. Staining for other B-cell, T-

TABLE II. Review of 18 Reported Cases of Follicular Dendritic Cell Tumors*

Report	Patient	Age (year)	Sex	Symptoms	Fever	Site of disease	Treatment	Response	Status	Follow-up (month)	Comment
Monda et al. [23]	1	40	F	LAD	NS	Cervical	Excision × 3	Rec	DOD	84	Treated for NHL
Monda et al. [23]	2	29	M	LAD	NS	Cervical	Excision	CR	ADF	12	Bone marrow negative
Monda et al. [23]	3	58	F	LAD	NS	Cervical	Excision/ chemo	LFU	?		Dx of NHL
Monda et al. [23]	4	38	M	LAD	NS	Cervical	Excision × 2/RoRx	Rec/CR	ADF	4	Dx of NHL and metastatic hemangioperi-cytoma
Pallesen and Myhre-Jensen [24]	5	39	M	LAD	NS	Axillary	Excision × 2	Rec/CR	ADF	90	Dx of MFH
Weiss et al. [19]	6	45	M	LAD	NS	Cervical	Excision	CR	ADF	12	
Weiss et al. [19]	7	66	F	LAD	NS	Axillary	Excision + RoRx	CR	ADF	27	
Hollowood et al. [25]	8	23	F	Pain	NS	Small bowel	Excision × 2	Rec/?	?		Last 2 months of pregnancy
Hollowood et al. [25]	9	63	M	WL	NS	Pancreas	Excision	CR	ADF	4	WL, nausea, epigastric mass
Chan et al. [22]	10	63	F	LAD	NS	Palate	RoRx/excision	CR	ADF	17	Initial Dx of acinic cell carcinoma
Chan et al. [22]	11	44	M	Polyp	NS	Tonsil	Tonsillectomy				No follow-up data available
Chan et al. [22]	12	43	M	Pain	NS	Mesenteric	Excision/ chemo	CR	ADF	12	Patient with Castleman's disease
Hollowood et al. [18]	13	49	F	Nodule	NS	Supra-clavicular	Excision × 2	Rec/CR	ADF	48	Dx of low-grade MFH
Hollowood et al. [18]	14	35	F	LAD	NS	Axillary	Excision	CR	ADF	6	No systemic disease
Tanda et al. [26]	15	31	F	LAD	NS	Sub-mandibular	Excision	Unknown	?		Previously diagnosed with Castleman's disease
Nguyen et al. [27]	16	60	F	LAD	NS	Cervical	Excision	CR	ADF	7	
Shek et al. [21]	17	35	F	Pain	Yes	Liver	Excision × 2	Rec/CR	?		Dx of hepatocellular carcinoma
This study	18	64	M	Pain	Yes	Liver, spleen	Chemo/ excision	PR/PR	AWD	10	Initial Dx of NHL

*ADF, alive disease-free; AWD, alive with disease; Chemo, chemotherapy; CR, complete remission; DOD, dead of disease; Dx, diagnosis; LAD, lymphadenopathy; LFU, lost to follow-up; MFH, malignant fibrous histiocytoma; NHL, non-Hodgkin's lymphoma; NS, not stated; PR, partial remission; Rec, recurrence; RoRx, radiotherapy; WL, weight loss.

cell, or Hodgkin's-associated markers was negative. No clonal T-cell receptor or immunoglobulin gene rearrangements were found.

Although most of the reported cases of FDCS have had primary lymph node involvement, the present case represents the second description of major liver involvement [21]. Such extranodal disease localization has been noted in the liver [21], oral cavity [22], pancreas, small bowel [25], and mesentery [20]. One of the patients with intra-abdominal disease presented with pain during the last 2 months of pregnancy and was found to have a 6.5-cm mass in the wall of the small bowel. In another patient, a mass in the head of the pancreas was resected, and no evidence of recurrence was detected at the latest follow-up.

Sarcomas derived from the IDRCs may be difficult to differentiate from FDCS [28,29]. Although morphologically similar, immunostaining can be helpful in establishing the correct diagnosis. IDRC sarcomas are usually positive for S-100 protein and negative for complement receptors, including CD21. The prognosis of IDRC sarcoma appears to be worse.

TABLE III. Results of Immunohistochemical Studies in 18 Reported Cases of Follicular Dendritic Cell Tumors*

	Patient	R4/23	Ki-M4	BU-10	CD35	CD11a	CD11b	CD11c	CD14	CD45	CD19	CD20	CD21	CD22	CD23	Vimentin	S-100 protein	Ki-67, %	HLA-DR
Monda et al. [23]	1	1			1					1						0	0		
Monda et al. [23]	2	0			0					1						0	0		
Monda et al. [23]	3															0	0		
Monda et al. [23]	4	1			1					1						0	0		
Pallesen and Myhre-Jensen [24]	5	0	1	1	1	1	0	0	0	0	1	1	1	0	1	1	0	5	1
Weiss et al. [19]	6	1			1			0	1	0	1		1	1			1	<5	1
Weiss et al. [19]	7	0			1			0	1	0			1	0			0	10	1
Hollowood et al. [25]	8				1					0		0	1				0		1
Hollowood et al. [25]	9				1					0		0	1				0		0
Chan et al. [22]	10				1					0		0	1				0		
Chan et al. [22]	11				1					1		0	1				0		
Chan et al. [22]	12				1					0		0	1				1		
Hollowood et al. [18]	13									0			1			0	0		0
Hollowood et al. [18]	14									0			1			0	0		0
Tanda et al. [26]	15				1								1						
Nguyen et al. [27]	16		1							0		0				0		0	
Shek et al. [21]	17	1	1		1					1	0		1	0	0	1	0	50	
This study	18								1	1		0		0	0	1	1		
No. tested		7	3	1	13	1	1	3	4	16	3	8	12	5	3	10	16	5	7
% tested		41	18	6	76	6	6	18	22	89	18	44	71	28	17	56	89	24	41
No. positive		4	3	1	12	1	0	0	3	6	2	1	12	1	1	3	3		4
% positive		57	100	100	92	100	0	0	75	38	67	13	100	20	33	30	19		57

*0 = no positive staining; 1 = stained positively.

IPT, a benign neoplasm of FDCs, usually presents with lymphadenopathy [30], fever, or weight loss, or some combination of these [31,32]. Both nodal and extranodal disease have been reported [16,17,33–35]. Although the morphological appearance of IPT [31] may be similar to that of FDCS, the lack of morphological atypia and aggressive growth pattern is a useful discriminatory feature. It has been postulated that FDCS may represent the malignant counterpart or the progression of IPT to a malignant tumor [26].

Hodgkin's disease, a tumor with possible FDC origin [36,37] should also be considered in the differential diagnosis [15]. IPT and spleen involvement were diagnosed in a patient in the process of a staging laparotomy for lymphocyte-predominance Hodgkin's disease [38]. Hodgkin's disease is characterized by positive staining for CD15 and CD30 antigens. Expression of the Epstein-Barr virus-related proteins has been demonstrated in histologic samples from patients with Hodgkin's disease [39], FDCS [21], and IPT [40]; we were unable to find Epstein-Barr mRNA in the neoplastic cells from our patient.

Other neoplastic entities such as anaplastic CD30 (Ki-1)-positive lymphoma [41] can have a spindle cell morphology similar to that of FDCS. In contrast to FDCS, this lymphoma displays the characteristic staining for CD30 (Ki-1) and has the specific chromosomal translocation t(2;5)(p23;q35). Kikuchi's disease, a rare lymphadenopathy of unknown cause, is usually differentiated from FDCS by its characteristic morphological appearance [42]. Intranodal myofibroblastoma, another tumor with spindle cell morphology [43], almost always presents with inguinal lymphadenopathy [44,45] and has characteristic amianthoid fibers.

Thus, FDCSs may present a diagnostic challenge to surgical pathologists. It may be difficult, on morphological review alone, to differentiate these tumors from other neoplastic processes, including Hodgkin's and non-Hodgkin's lymphomas and soft tissue sarcomas. Therefore, it is imperative to complement the morphological review with immunophenotypic and genetic studies. It should be noted that the initial histologic diagnosis in our patient was reviewed by a panel of hematopathologists and reported as large cell lymphoma. Only after standard lymphoma chemotherapy failed was the patient reevaluated and additional biopsy results and immunohistochemical analyses confirmed the diagnosis of an FDCS. Similarly, in several previously reported cases, the initial diagnosis was something other than FDCS, for example, lymphoma [23], malignant fibrous histiocytoma [18,24], and metastatic hemangiopericytoma [23]. Our patient exemplifies some of the usual complexities involved in the diagnosis and treatment of FDCS.

REFERENCES

1. Heinen E, Tsunoda R: Microenvironments for B-cell production and stimulation. Immunol Today 8:142, 1987.
2. Heinen E, Coulie P, Van Snick J, Braun M, Cormann N, Moeremans M, Kinet-Denoel C, Simar LJ: Retention of immune complexes by murine lymph node or spleen follicular dendritic cells. Role of antibody isotype. Scand J Immunol 24:327, 1986.
3. Gerdes J, Stein H, Mason DY, Ziegler A: Human dendritic reticulum cells of lymphoid follicles: Their antigenic profile and their identification as multinucleated giant cells. Virchows Arch B Cell Pathol Incl Mol Pathol 42:161, 1983.
4. Szakal AK, Gieringer RL, Kosco MH, Tew JG. Isolated follicular dendritic cells: Cytochemical antigen localization, Nomarski, SEM, and TEM morphology. J Immunol 134:1349, 1985.
5. Friess A: Interdigitating reticulum cells in the popliteal lymph node of the rat. An ultrastructural and cytochemical study. Cell Tissue Res 170:43, 1976.
6. Johnson GD, Hardie DL, Ling NR, Maclennan IC: Human follicular dendritic cells (FDC): A study with monoclonal antibodies (MoAb). Clin Exp Immunol 64:205, 1986.
7. Parwaresch MR, Radzun HJ, Hansmann ML, Peters KP: Monoclonal antibody Ki-M4 specifically recognizes human dendritic reticulum cells (follicular dendritic cells) and their possible precursor in blood. Blood 62:585, 1983.
8. Petrasch S, Perez-Alvarez C, Schmitz J, Kosco M, Brittinger G: Antigenic phenotyping of human follicular dendritic cells isolated from nonmalignant and malignant lymphatic tissue. Eur J Immunol 20: 1013, 1990.
9. Schriever F, Freedman AS, Freeman G, Messner E, Lee G, Daley J, Nadler LM: Isolated human follicular dendritic cells display a unique antigenic phenotype. J Exp Med 169:2043, 1989.
10. Sellheyer K, Schwarting R, Stein H: Isolation and antigenic profile of follicular dendritic cells. Clin Exp Immunol 78:431, 1989.
11. Ralfkiaer E, Stein H, Plesner T, Hou-Jensen K, Mason D: In situ immunological characterization of Langerhans cells with monoclonal antibodies: Comparison with other dendritic cells in skin and lymph nodes. Virchows Arch A Pathol Anat Histopathol 403:401, 1984.
12. Mandel TE, Phipps RP, Abbot A, Tew JG: The follicular dendritic cell: Long term antigen retention during immunity. Immunol Rev 53: 29, 1980.
13. Klaus GG, Humphrey JH, Kunkl A, Dongworth DW: The follicular dendritic cell: Its role in antigen presentation in the generation of immunological memory. Immunol Rev 53:3, 1980.
14. Tew JG, Kosco MH, Burton GF, Szakal AK: Follicular dendritic cells as accessory cells. Immunol Rev 117:185, 1990.
15. Davis RE, Warnke RA, Dorfman RF: Inflammatory pseudotumor of lymph nodes. Additional observations and evidence for an inflammatory etiology. Am J Surg Pathol 15:744, 1991.
16. Matsubara O, Tan-Liu NS, Kenney RM, Mark EJ: Inflammatory pseudotumors of the lung: Progression from organizing pneumonia to fibrous histiocytoma or to plasma cell granuloma in 32 cases. Hum Pathol 19:807, 1988.
17. Shek TW, Ng IO, Chan KW: Inflammatory pseudotumor of the liver. Report of four cases and review of the literature. Am J Surg Pathol 17:231, 1993.
18. Hollowood K, Pease C, Mackay AM, Fletcher CD: Sarcomatoid tumours of lymph nodes showing follicular dendritic cell differentiation. J Pathol 163:205, 1991.
19. Weiss LM, Berry GJ, Dorfman RF, Banks P, Kaiserling E, Curtis J, Rosai J, Warnke RA: Spindle cell neoplasms of lymph nodes of probable reticulum cell lineage. True reticulum cell sarcoma? Am J Surg Pathol 14:405, 1990.
20. Chan JK, Tsang WY, Ng CS: Follicular dendritic cell tumor and vascular neoplasm complicating hyaline-vascular Castleman's disease. Am J Surg Pathol 18:517, 1994.

21. Shek TWH, Ho FCS, Ng IOL, Chan ACL, Ma L, Srivastava G: Follicular dendritic cell tumor of the liver: Evidence for an Epstein-Barr virus-related clonal proliferation of follicular dendritic cells. Am J Surg Pathol 20:313, 1996.
22. Chan JK, Tsang WY, Ng CS, Tang SK, Yu HC, Lee AW: Follicular dendritic cell tumors of the oral cavity. Am J Surg Pathol 18:148, 1994.
23. Monda L, Warnke R, Rosai J: A primary lymph node malignancy with features suggestive of dendritic reticulum cell differentiation. A report of 4 cases. Am J Pathol 122:562, 1986.
24. Pallesen G, Myhre-Jensen O: Immunophenotypic analysis of neoplastic cells in follicular dendritic cell sarcoma. Leukemia 1:549, 1987.
25. Hollowood K, Stamp G, Zouvani I, Fletcher CD: Extranodal follicular dendritic cell sarcoma of the gastrointestinal tract. Morphologic, immunohistochemical and ultrastructural analysis of two cases. Am J Clin Pathol 103:90, 1995.
26. Tanda F, Massarelli G, Cossu A, Bosincu L, Canu L: Follicular dendritic cell sarcoma following inflammatory pseudotumor in pregnancy (abstract). Int J Surg Pathol 2:S283, 1995.
27. Nguyen DT, Diamond LW, Hansmann M-L, Hell K, Fischer R: Follicular dendritic cell sarcoma: Identification by monoclonal antibodies in paraffin sections. Appl Immunohistochem 2:60, 1994.
28. van Heerde P, Feltkamp CA, Feltkamp-Vroom TM, Koudstaal J: Sarcoma arising from interdigitating cells. Cytology and cytochemistry. Acta Cytol 27:306, 1983.
29. van den Oord JJ, de Wolf-Peeters C, de Vos R, Thomas J, Desmet VJ: Sarcoma arising from interdigitating reticulum cells: Report of a case, studied with light and electron microscopy, and enzyme- and immunohistochemistry. Histopathology 10:509, 1986.
30. Perrone T, De Wolf-Peeters C, Frizzera G: Inflammatory pseudotumor of lymph nodes. A distinctive pattern of nodal reaction. Am J Surg Pathol 12:351, 1988.
31. Facchetti F, De Wolf Peeters C, De Wever I, Frizzera G: Inflammatory pseudotumor of lymph nodes. Immunohistochemical evidence for its fibrohistiocytic nature. Am J Pathol 137:281, 1990.
32. Kemper CA, Davis RE, Deresinski SC, Dorfmann RF: Inflammatory pseudotumor of intra-abdominal lymph nodes manifesting as recurrent fever of unknown origin: A case report. Am J Med 90:519, 1991.
33. Pettinato G, Manivel JC, De Rosa N, Dehner LP: Inflammatory myofibroblastic tumor (plasma cell granuloma). Clinicopathologic study of 20 cases with immunohistochemical and ultrastructural observations. Am J Clin Pathol 94:538, 1990.
34. Anthony PP, Telesinghe PU: Inflammatory pseudotumor of the liver. J Clin Pathol 39:761, 1986.
35. Someren A: ''Inflammatory pseudotumor'' of liver with occlusive phlebitis: Report of a case in a child and review of the literature. Am J Clin Pathol 69:176, 1978.
36. Soderstrom KO, Rinne R, Hopsu-Havu VK, Jarvinen M, Rinne A: Hodgkin's disease: A malignancy of follicular dendritic cells? (Letter) Lancet 343:422, 1994.
37. Curran RC, Jones EL: Dendritic cells and B lymphocytes in Hodgkin's disease (letter). Lancet 2:349, 1977.
38. Cotelingam JD, Jaffe ES: Inflammatory pseudotumor of the spleen. Am J Surg Pathol 8:375, 1984.
39. Pallesen G, Hamilton-Dutoit SJ, Rowe M, Young LS: Expression of Epstein-Barr virus latent gene products in tumour cells of Hodgkin's disease. Lancet 337:320, 1991.
40. Arber DA, Kamel OW, Davis RE, Van de Rijn M, Weiss LM: Detection of the Epstein-Barr virus in inflammatory pseudotumor (abstract). Mod Pathol 7:102A, 1994.
41. Chan JK, Buchanan R, Fletcher CD: Sarcomatoid variant of anaplastic large-cell Ki-1 lymphoma. Am J Surg Pathol 14:983, 1990.
42. Dorfman RF, Berry GJ: Kikuchi's histiocytic necrotizing lymphadenitis: An analysis of 108 cases with emphasis on differential diagnosis. Semin Diagn Pathol 5:329, 1988.
43. Weiss SW, Gnepp DR, Bratthauer GL: Palisaded myofibroblastoma. A benign mesenchymal tumor of lymph node. Am J Surg Pathol 13:341, 1989.
44. Suster S, Rosai J: Intranodal hemorrhagic spindle-cell tumor with ''amianthoid'' fibers. Report of six cases of a distinctive mesenchymal neoplasm of the inguinal region that simulates Kaposi's sarcoma. Am J Surg Pathol 13:347, 1989.
45. Fletcher CD, Stirling RW: Intranodal myofibroblastoma presenting in the submandibular region: Evidence of a broader clinical and histological spectrum. Histopathology 16:287, 1990.